RAD7 encodes a protein that acts in nucleotide excision repairof UV-damaged DNA. In NER, damaged DNA is excised by incision of the DNA on either side of the lesion followed by unwinding by DNA helicases; the process of NER is reviewed in 5. Rad7p and Rad16p form a stoichiometric complex called NEF4. NEF4 binds specifically to UV-damaged DNA in an ATP-dependent manner, and has DNA-dependent ATPase activity. Rad4p and Rad23p form another complex, called NEF2, that binds cooperatively with NEF4 to damaged DNA. Unlike NEF2, NEF4 is not required for incision, but the in vitro incision reaction is enhanced when both NEF2 and NEF4 are present. In the absence of Rad16p, Rad7p interacts physically with NEF2, and binds DNA but does not show specificity for damaged DNA. rad7 mutants, like rad16 mutants, are proficient in incision but defective in both oligonucleotide excision and repair synthesis, suggesting that Rad7p and Rad16p act in a post-incision step of NER.In humans, NER deficiencies are associated with xeroderma pigmentosumand Cockayne syndrome. Homologs of Rad7p and Rad16p have been identified in S. pombe.